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"It is of little moment whether one studies inflammation in the periodontal tissue or in some other part of the body. The principles involved are exactly the same . . .

"It is interesting to note what happens in the initial stage when any sort of irritant is introduced into an otherwise normal tissue. There is at first a disturbance in fluid exchange. This is referable to a transitory increase in capillary pressure; but of greater importance is the enhancement in capillary permeability which accompanies any form of inflammation. This lasts throughout a large period of the inflammatory reaction, and it is probably the most important of initial changes in the development of inflammation. Inasmuch as all later sequences in the so-called inflammatory reaction depend upon this pivotal reaction of increased capillary permeability, it would be well for us to discuss the mechanism involved."

Valy Menkin, M.D.
See p. 166

THE JOURNAL OF PERIODONTOLOGY

OCTOBER. NINETEEN HUNDRED FORTY-SEVEN

Diabetes Mellitus, Its Relation to Oral Pathosis: A Symposium*

I. PATHOLOGICAL CHANGES IN EXTRA-ORAL TISSUES.

By ALEXANDER MARBLE, M.D., *Boston, Massachusetts***

THERE are at least a million diabetics in the United States today and recent studies (1) suggest that there may be twice that number. About 1 in 4 persons in the population has a diabetic relative. Diabetes now ranks eighth as a cause of death in the country and this does not take into account those diabetics dying of other causes which take precedence on the death certificate. It is thus an important disease which directly or indirectly affects the lives of many people. Its consideration cannot fail therefore to be of value to any medical group.

Diabetes mellitus is a metabolic disease which, if uncontrolled, may exert a deleterious effect on every tissue and organ in the body. The present paper will be limited to abnormalities noted in tissues other than those about the mouth. It will be convenient to divide the discussion into two parts, mentioning first those changes of acute nature noted relatively early in poorly controlled or untreated diabetes and second, those of a more chronic nature seen only after months of incomplete control. Unless the diabetes is severe enough or the degree of poor control marked enough, certain of these pathological changes may not become evident clinically until after years have passed. This element of time is an important consideration to which further reference will be made later. In general, the changes of acute nature may be regarded as reversible whereas those of chronic nature are irreversible. It should be emphasized, however, that there is no sharp dividing line between the early and the late effects; with long-continued poor control, the acute abnormalities lead up to and merge with those of more chronic nature.

EARLY, REVERSIBLE CHANGES

The classical symptoms of diabetes—polydipsia, polyuria, polyphagia, pruritus, and loss of weight and strength—are well known, although it should be noted that no symptoms whatever may be present. These symptoms may be associated with abnormal signs found on physical examination, chiefly dehydration and evidence of loss of weight together with diminished muscular power and lack of endurance. In the diabetic, food which is eaten is digested in presumably

*Presented at the Annual Meeting of the American Academy of Periodontology, *Boston, Mass.*, July 31, 1947.

**From the George F. Baker Clinic, Elliott P. Joslin, M.D., Medical Director, New England Deaconess Hospital, *Boston*.

normal fashion, but the end-products of digestion when absorbed into the body proper are poorly utilized, i.e., they are neither used in normal fashion for heat and energy nor are they properly stored in the liver, muscles, and skin for future use. Instead, glucose resulting both from the ingestion of food and from the excessive breakdown of glycogen, increases the content of sugar of the body fluids generally. This is reflected in an abnormally high content of glucose in the blood with an overflow and wastage of sugar in the urine.

The blood sugar of the severe diabetic is abnormally high even in the fasting state due to the breakdown of glycogen stores just mentioned, presumably under the influence of secretions of the pituitary and adrenal glands which are regarded as the normal antagonists of insulin in the body's scheme to provide homeostasis. As the result of this wasting of food, both ingested and stored, the glycogen reserves of the body become depleted. In this emergency, the body calls in excessive fashion upon other stores, namely, those of fat and to some extent, of protein. In everyday life protein is broken down only to the extent necessary for body activities and although fat is used normally for energy, carbohydrate is used preferentially. In severe, uncontrolled diabetes when adequate carbohydrate is no longer available, more fat is broken down than can be used by the tissues. As a consequence, the end-products of fat breakdown, the so-called acetone bodies, are formed in great excess and flood the organism. Acidosis is then said to be present and, if unchecked, this leads eventually to definite coma. It is understandable that in diabetic coma one finds in the blood not only an excess of sugar but also the presence of acetone bodies and a lowered content of carbon dioxide. In the urine one finds sugar, acetone, and an increased amount of nitrogen reflecting the excessive protein destruction. All of the above abnormalities are correctible if the patient receives early enough an adequate amount of insulin together with sufficient fluid and food. The changes therefore fall into the category of acute, reversible processes.

Not all of the acute changes seen in uncontrolled diabetes are as dramatic as those seen in frank acidosis. It is not necessary here to discuss them in detail but they include the complications listed below.

(1) Blurring of vision due to myopia brought about by changes in the lens of the eye with dehydration;

(2) A tendency to the development of skin lesions, as (a) boils and carbuncles, due presumably to lowered resistance, although the exact nature of the mechanism involved is not clear and, less commonly, (b) xanthomatous lesions accompanying an increased content of cholesterol in the blood;

(3) Enlargement of the liver, presumably due to fat, seen particularly prior to the development of depot insulins, such as protamine zinc insulin, in children and adolescents with severe diabetes without which it is difficult to accomplish anything approaching continuous control of diabetes;

(4) Muscle cramps and pains down the extremities, especially the legs. This last-named effect deserves further consideration for it may at times amount to a true diabetic neuritis which, although almost invariably traceable to a period of poor control, may not respond readily to treatment with diet, insulin and vitamin B but instead requires weeks and even months for relief. Indeed, so long-lasting may be the period of disability of diabetic neuritis that one may question the propriety of including it in the list of acute complications. However, in almost every instance, with meticulous treatment the condition is found to be reversible, at least in the sense that the patient eventually recovers from the severe neuritic pains, paraesthesias, weakness of muscles, and even paralyses which over such a prolonged period have been disabling.

To the above group of complications must be added certain abnormalities which if recognized and treated early enough may be overcome. Reference is made particularly to the retardation of growth and development formerly seen in certain children with severe diabetes, particularly those with onset of the disease under the age of 5 years. In addition to being underheight for age with delay of osseous development, these retarded children or diabetic dwarfs show immature behavior, protuberance of the abdomen with enlargement of the liver and delay of sexual development. Since the advent of protamine zinc insulin, diabetic dwarfs are seen only infrequently. Treatment consists in careful control of the diabetes, provision for a diet with an abundance of protein and adequate in calories and in selected cases the use of thyroid extract, pituitary extract, and the sex hormones, testosterone and estrogen. It

is obvious that acceleration of growth can be hoped for only in those patients whose bone epiphyses are still open.

Mention should be made of the difficulty which pregnant women who have had diabetes since childhood or adolescence experience in securing a living baby. Even in the insulin era, fetal mortality has until a decade ago been no lower than 40 to 50 per cent, i.e., 4 or 5 of every 10 pregnancies ended unsuccessfully. In the last 10 years Dr. Priscilla White (2) has achieved remarkable results by means of constant, close supervision of the patient during pregnancy and particularly by the use of hormone therapy, including large doses of estrogens and progesterone. In patients in whom studies have shown hormonal imbalance, she has been able to secure by treatment more than 90 per cent fetal survival as compared with 50 per cent in an earlier untreated group.

IRREVERSIBLE, DEGENERATIVE CHANGES

Distressing though the acute, reversible effects of uncontrolled diabetes may be, they are not—apart from coma and long-continued acidosis—to be compared in seriousness with the chronic, irreversible, degenerative changes which follow a long period of poorly controlled diabetes. Actually, of course, the irreversible effects follow persistence of the conditions of poor control which for a time are manifest only by the acute difficulties just discussed. Here must be emphasized the element of time mentioned at the beginning of this paper for many have been misled in their thinking because of failure to recognize that the degenerative complications of diabetes develop only gradually over 5, 10, 15 or 20 years of time. Some have said in recent years that hyperglycemia and glycosuria are not harmful as long as the patient appears in good health and is free from symptoms. However, the writer and his associates believe that long-continued hyperglycemia and glycosuria *are* harmful if only as indices of poorly controlled diabetes, granted that at the present time no one knows exactly what it is about poorly controlled diabetes which results in the degenerative complications. Added to this is the recent work of Dohan and Lukens (3) in which it was shown that permanent diabetes may be produced in the normal cat by the artificial production and maintenance of long-continued hyperglycemia.

What, then, are the degenerative complications of diabetes and in what patients may one best study them? They are chiefly vascular in nature and may be best observed in patients with severe diabetes with onset in childhood and duration of 10, 15 or 20 years. Experience has shown that incompletely controlled diabetes predisposes to hardening of the arteries and that arteriosclerosis occurs prematurely and to a greater degree in diabetics of all ages as compared to non-diabetics. However, recognizable arteriosclerosis occurs eventually to a greater or less degree in almost all persons reaching middle age. Furthermore, older individuals are subject to other consequences of aging so that it is not easy to draw definite conclusions from a study of middle-aged or elderly people. Consequently, one turns preferably to diabetics with onset of the disease in childhood for here the process is seen in its purest, and often most severe, form. The occurrence of well-marked arteriosclerosis in persons in their 20s, 30s and 40s must be accepted as a finding of unquestioned significance.

Our own series of patients with onset of diabetes at age 15.0 years or under, studied over years of time by Dr. Priscilla White, comprised up to January 1946, 2191 cases, 1774 of whom were living and 385 dead, with 32 untraced. Of these, 249 had survived 20 or more years of diabetes. An appreciable number of these have been studied by Dr. White (4) for evidences of arteriosclerosis and its sequelae in the favorite sites, the eyes, heart, kidneys and peripheral vessels. The studies have included physical examination including examination of the optic fundi, evaluation of renal function, electrocardiograms, X-rays of the chest, and X-rays of the peripheral vessels, pelvis, and aorta to demonstrate possible sclerosis. The findings as analyzed by Dr. White show that at least 70 per cent have definite evidence of arteriosclerosis after 20 years of diabetes. Clinical findings include retinal sclerosis, often accompanied by retinitis (and at times retinitis proliferans) leading to partial or complete blindness; arteriosclerotic heart disease with coronary sclerosis and angina; hypertension; sclerosis of the aorta and peripheral vessels with diminished circulation in the extremities leading at times to gangrene and the necessity for amputation of a part of a foot or leg; sclerosis of the pelvic vessels carrying with it a poor prognosis for a successful termination of pregnancy; and worst

of all, chronic nephritis often of a special type called intercapillary glomerulosclerosis with marked albuminuria, greatly diminished renal function, and, in a certain stage, the development of nephrosis with a markedly lowered content of protein in the blood plasma, reversal of the albumin-globulin ratio, azotemia and generalized edema. In the period from 1941 to 1946, nephritis became the chief cause of death in patients with onset of diabetes in childhood with 26 of 76 deaths due to this complication.

It is not only in erstwhile childhood diabetics but also in diabetics of all age groups that arteriosclerosis with its sequelae has become the major complication and the chief enemy, now trading places with diabetic coma which took such a devastating toll in the days before insulin. With insulin diabetic coma can be prevented and treated with success so that as the years have gone by since 1922, the mortality from coma has become less and less, accounting now in our own cases for less than 3 per cent of all deaths. With insulin and with the use of sulfonamides, penicillin, and streptomycin, deaths from infections have diminished progressively. Deaths from cancer do not form a large percentage of the total deaths. One is left with the fact that diabetics become older and live longer with their disease and as the other important complications have been overcome, deaths from arteriosclerosis whether in the heart, extremities, kidneys or brain have increased, so that in the latest tabulation of our cases 67 per cent of diabetic deaths were due to arteriosclerosis.

Reference already has been made to retinitis as an important complication. Fine, deep retinal hemorrhages may be an early sign of vascular damage in the body. The exact cause is not clear although undoubtedly retinitis follows poor control of diabetes. Recent studies have shown that in most patients with retinitis there is an increase in capillary fragility. The latter may often be brought to normal with the use of the newly introduced drug known as rutin but despite this, the retinitis may not be appreciably influenced. More work is needed before definite conclusions may be drawn. The retinitis in diabetics unfortunately may be extensive and progressive and lead to near or total blindness. Retinitis proliferans is a not infrequent complication.

Finally, there are a group of complications of irreversible nature which may be grouped under the head of diabetic neuropathies. Earlier in the paper reference has been made to one manifestation of the effect of diabetes on the nervous system, namely, diabetic neuritis. Although, as stated, this is usually in time a remediable condition, there are neuropathies in which treatment thus far has proved to be of only slight benefit. These include: (a) Neuropathic foot, a condition of infrequent occurrence in which changes occur in the tarsal and metatarsal bones similar to those found in the Charcot joint of syphilis. Slow progression of the destructive processes leads to a thickened, deformed foot with a tendency to eversion, external rotation, and a flattening of the longitudinal arch; (b) Neurogenic or "cord" bladder, in which paralysis of the urinary bladder occurs followed frequently by urinary tract infection; (c) "Diabetic diarrhea," may possibly belong under the heading of neuropathies. This condition, without discoverable cause except for poorly controlled diabetes, is an infrequent but distressing complication in which the diarrhea is often nocturnal. Although in most cases symptomatic relief may be obtained by the parenteral use of crude liver extract, cures are uncommon and relapses usually follow cessation of treatment (5).

COMMENT

Enough has been said to indicate the widespread influence which diabetes has upon the body. No tissue is exempt and the complications are of such nature as to demand respect. However, there is a much brighter side to the story and it would not be fair to conclude without emphasizing these hopeful and happier aspects of diabetes as seen today. Throughout the preceding discussion it has been repeatedly mentioned that this or that complication is the result of uncontrolled or poorly controlled diabetes. Conversely, with excellent control of diabetes, these distressing complications can be, and are, in large part avoided or delayed. Careful adherence to an adequate yet restricted diet, together with the use of insulin in truly adequate dosage, yields the reward of health and relative freedom from complications. It is common knowledge that before insulin it was the unusual child who lived for 2 years after the onset of diabetes; now diabetic children are living on and on after 20 or more years of the disease. As

already stated, in our own group alone there were up to January 1946, 249 patients with onset in childhood who had survived 20 or more years of diabetes; at the time of tabulation only 12 of these had died. The average diabetic is a middle-aged or elderly person; he now lives an essentially normal life. Whereas in the period from 1898 to 1914 he died at the age of 44.5 years after 4.9 years of diabetes, he now lives to the age of 64.5 years after 14.1 years of diabetes. The life expectancy of the diabetic with onset of the disease in middle life now approaches, although it does not quite equal, that of the non-diabetic of comparable age. The man-years saved by insulin in the last quarter-century for the productivity of the world in all occupations and professions is enormous. On the basis of this achievement one can anticipate even greater advances in the future, hopefully in the field of prevention.

SUMMARY

1. Reversible complications of diabetes include acidosis, certain visual disturbances, skin lesions, neuritis, retardation of growth and development, and increased fetal mortality in diabetic pregnancies.

2. Irreversible, degenerative complications include arteriosclerosis affecting the eyes, heart, kidneys, brain and peripheral vessels, retinitis, and neuropathies of various types.

3. The complications listed reflect incompletely controlled diabetes and can be avoided only if careful, continuous treatment is provided.

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II. ORAL LESIONS SEEN IN DIABETES MELLITUS*

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AS A PHYSICIAN it is with a good deal of hesitation that I talk to you as dentists. The classical request "stick out your tongue please" followed by a cursory look at this organ was the extent of the oral examination by the physician. If the patient had complaints referable to the mouth, the physician in all confidence dismissed them by referring the patient to the dentist. The dentist began referring the patients with increasing frequency back to the physician. He was able to interpret the indicator pathology and suggest that there might be systemic disease. Usually he was correct. Today the closer relationship between the two is of greater importance than ever.

Physicians interested in metabolic and nutritional disturbances now examine the mouth before they take a blood pressure reading. A summary of the nutrition surveys, conducted here and abroad, reveals that over half the examination is conducted in and around the mouth. The lips, the tongue, the gingiva, and the buccal mucous membrane indicate early deficiencies in iron, riboflavin, nicotinic acid, and other essential nutrients. It is you as dentists who frequently see these lesions first. It is you who

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must learn to recognize them and see to it that the patient gets proper therapy.

Today the oral lesions in that metabolic disease, diabetes mellitus, are the subject of discussion. The obvious question to be answered is, "Are there any specific lesions of diabetes seen in the mouth?" The answer is not as simple as the question. The diabetic is subject to all the lesions seen in the non-diabetic. Their gross appearance may be altered because of the presence of the diabetes, but their underlying pathology is the same.

Diabetes mellitus is not a disease entity. It is a clinical syndrome characterized by hyperglycemia and glycemia. Years ago jaundice was listed as a disease. Today one knows that there are different types of jaundice and each type has numerous causes. Clinically one can differentiate many types of diabetes. The juvenile type is quite different from the diabetes that has its onset after fifty years of age. The diabetes of hyperthyroidism, Cushing syndrome, hepatic disease, and obesity are all different in their development, and course. The diabetes of total pancreatectomy in the human is very different from any of the spontaneous diabetes that one sees clinically. One would imagine that total pancreatectomy in humans would produce a severe type. On the contrary, it is usually mild, very stable, easy to control, and never requires more than 40 to 50 units of insulin daily, usually less. When this concept is brought out, it is not remarkable that the oral lesions described in diabetics should be so varied. The lesions will vary with the type of patient, the age, duration of the diabetes, cause of the diabetes, and what other nutritional or metabolic conditioning factors are present.

The older experiences with diabetes as described in the literature, and as many of us remember them, may not bear a close relationship to present day experience. The history of diabetes falls into several eras. The pre-insulin, (before 1921), the Banting era from 1921 to 1938, and the protamine zinc insulin era since 1938. The results in the therapy of this syndrome have changed with each of these eras. Prior to the introduction of insulin the juvenile diabetic survived on an average of 1 year. He did not have a chance to develop the mouth changes which require longer periods of time. In the Banting era these patients were kept alive, but the majority were not kept in anything like an approximation of the physiological normal. It was in these eras that so many oral lesions of diabetes were described. With the introduction of protamine zinc insulin there was a definite improvement in the results of therapy. The ease with which a patient could be kept in a positive nitrogen balance improved the physiological status of the patient. Under this therapy the type of the oral lesions changed and the frequency decreased. It will, however, take years before the full impact of this improved therapy can be finally evaluated.

In the literature one finds the following conditions, singly and in combination, described as being characteristically found in diabetic patients:

Sore tooth (teeth), periodontitis, sensitivity to percussion, gingival abscess, loose teeth, alveolar resorption not due to local causes, foul breath, thirst and dryness of mouth, fissured tongue, swelling of tongue —enlarged and discolored papillae, stomatitis, gingivitis, hypertrophy and inflammation of gingival papillae, gingival bleeding, recession of gingivae and pocket formation, paradentosis, periodontoclasia, pyorrhoea alveolaris, sensitive cementum, circular caries, thrush and a peculiar violaceous color of gums in juvenile diabetes. Also thicker gums and increased crevicular depth in young diabetics.

These lesions were described by Niles (1), Rudy and Cohen (2), Lyons (3), Sheppard (4), Aiguier (5), Zilz (6), Williams (7), Schonfelder (8), Ziskin (9), *et al.* Most of these, one can dismiss as coincidental findings somewhat altered by the underlying abnormal physiology. It is true that the uncontrolled diabetic is more susceptible to any infection. An infection once incubated, spreads more rapidly in the diabetic than in the non-diabetic. The gross appearance in the mouth, will then be quite different. It is the virulence of the gingival abscess rather than the abscess itself which is the result of the diabetes. Thus the dentist can suspect diabetes by the extent and rapid spread of any mouth infection.

Rudy and Cohen (2) state, "The paradentium of patients with untreated or inadequately controlled diabetes presents a characteristic picture, loosening of the teeth and pain, hypertrophied inflamed gingival papillae; bleeding and pain of the gingiva and paradentium, gingival abscess; and excessive polypoid proliferation growing from

under the free margin of the gum", Sheppard (4) states that there is a high incidence of unusual resorption (of the alveolar bone) between the ages of 15 and 40 in diabetic patients, which can hardly be attributed to local causes and which is not found in healthy persons. Williams (7) noted that teeth were loose, gums hypertrophied, inflamed, dark red, ulcerated, and colored in spots with grayish areas of ulcerated tissue.

In the past, several reports have appeared indicating that dental caries experience in controlled diabetics differed from that of other population samples (2) (11) (12) (13) (14). More recent reports, however, using more refined statistical methods tend to invalidate this concept, and several of the authors have revised their opinions. It would appear that today, the generally accepted belief is that diabetics do not differ significantly from any other individuals as regards dental caries experience (10) (15) (16).

Periodontal disease thus has been repeatedly reported as present in diabetes mellitus. It is the same periodontal disease as seen in non-diabetics, but with these exceptions. It appears earlier and progresses more rapidly than in the non-diabetic. It is not present in all diabetics. The probable reason is that this rapid bone destruction is characteristic of only one of the metabolic anomalies associated with hyperglycemia and glycosuria.

Periodontal disease refers to disease in any of the supporting tissues of the teeth. These are the cementum, periodontal membrane with its nerves, blood vessels and lymphatics, alveolar bone, and (alveolar) gingivae. All of these are bound together by a dense collagenous fibrous tissue.

You are all familiar with the three main concepts of the etiology of the breakdown of the periodontal structures. The first indicates that the process is purely local in origin. Such factors as faulty hygiene, impaired function, and traumatic occlusion are considered the etiological agents. The second hypothesis puts the onus on systemic factors. These control the rates of bone formation and bone resorption, a dynamically continuous process normally. Periodontoclasia [periodontal disease] is said to result when bone resorption exceeds bone formation. The third concept is briefly a combination of the first and second. Clinically one can see types which can be used to substantiate any one of the three theories. Differential diagnosis is often very difficult. Only an accurate history from an intelligent patient will sometimes enable one to say whether the pocket formation and infection were secondary to shifting of the teeth from severe bone loss or whether the bone loss was secondary to the infection extending down Volkmann's canals into the alveolar bone. As stated previously, diabetes has been frequently implicated in this disease. One often sees extensive bone resorption without evidence of infection, primary or secondary, justifying the concept of a systemic factor.

Ziskin and his associates (17) studied sections of gingival tissue from insulin and diet controlled diabetics. They found hyperkeratinization, hyperplasia of epithelium and of connective tissue, increased glycogen deposition, increase in fibroblasts and prominence of the capillary bed with reduction of inflammatory exudate in the corium. They discuss the relationship of the changes noted to nutritional and metabolic disturbances which may exist and say, "The underlying factors in producing the gingival changes in the diabetics may be the nutritional and metabolic imbalances which the insulin treatment or diet only partially corrects". The increase in the capillary bed, and reduction of inflammatory exudate was thought to be due to the action of insulin, and of possible importance in combating infection.

Glickman (18) has performed a detailed study of changes in the periodontium in alloxan diabetes in rats. His findings were:

"(1) No gross pathological changes were noted in either control or diabetic animals.

"(2) The similarity in the microscopic appearance of the gingival mucosa, interdental gingival papilla, and gingival crevices of control and diabetics was striking. No changes were observed in those areas in diabetic animals which did not appear with comparable frequency and severity in the control group.

"(3) 39 per cent of the diabetic animals presented pathological bone changes. These varied in individual animals and were not correlated with the degree of hyperglycemia, or pancreas disturbance. It was found that a tendency toward varying degrees of nonspecific osteoporosis of alveolar bone unrelated to gingival changes is a feature in individual cases of diabetes mellitus.

"(4) It was further found that when

marked pathological changes occur throughout alveolar bone in diabetic animals, comparable alterations are observed in other bones of the skeletal system."

The final explanation for these rapid bone changes in certain types of diabetes is not yet available. A possible explanation may be offered. The acute and uncontrolled diabetic is usually in a negative nitrogen balance. This was particularly true in the pre-protamine zinc insulin days. This negative nitrogen balance means that there is a drainage of the body proteins. Calcium metabolism and its transport through the blood are dependent upon an adequate protein metabolism. Thus it is conceivable that with depleted proteins, there is a breakdown in the balance between bone formation and bone resorption. This same disturbance in the protein metabolism of the diabetic may explain his susceptibility to infection. It is known that the immune bodies in the blood are in the gamma globulins. In starvation and diabetes, the gamma globulins may disappear from the blood and, hence, the loss of resistance to infection.

Regardless of the specificity of any of these lesions, one thing can be stated with certainty. Symptomatic control of the diabetes with a proper diet to insure full nutritional rehabilitation, and protamine zinc insulin to correct the metabolic abnormalities, will do much to prevent these lesions. Certainly no surgical procedure can be successful unless there is simultaneous control of the diabetes. The criteria for the control of diabetes today have in the minds of some become somewhat distorted. Good control means the approximation of the physiological normal. It is not normal to have an elevated blood sugar concentration or to excrete sugar in the urine. The patient must consume an adequate diet to supply his caloric and specific nutrient requirement. He must have adequate insulin to enable him to metabolize this diet. The only time we compromise with these criteria is when the patient is subject to frequent and severe insulin reactions. Then one must investigate the cause of these reactions and remedy it as soon as possible.

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Blood Dyscrasias As Related to Periodontal Disease: With Special Reference to Leukemia*

By THOMAS J. COOK, D.D.S., *Miami, Florida*

INTRODUCTION

IN 1926 PRINZ[1], in a paper before the New York Dental Society, said "the non-surgical diseases of the oral cavity have, comparatively speaking, received only scant attention from the medical practitioner and, as a consequence, many of the more frequently occurring disturbances, i.e., primarily those of the mucous membrane, have by necessity gradually drifted into the hands of the dentist". He felt that it was therefore rational to assume that the dentist, by virtue of his specific training should be the logical practitioner to whose domain the treatment of intraoral lesions should be entrusted.

In 1927, at the annual meeting of this Academy the challenge was accepted in an excellent report by a member of your committee on Scientific Investigation, I. Hirschfeld[2], who wrote as follows:

As specialists in periodontia, the members of this Academy are not infrequently called on to diagnose and treat very unusual and severe periodontal diseases or periodontal symptoms of systemic disease. Yet it will be admitted that, up to the present time, aside from the stereotyped conditions that are found in our textbooks, comparatively little has been taught in our medical or dental schools to aid us in this direction. Both professions are still quite in the dark regarding many pathologic phenomena that present themselves in the mouth, especially those involving the gums. It is not strange, then, that the periodontist is often in a quandary as to proper diagnosis and treatment, and even medical aid cannot at times help him in solving the clinical puzzle pictures. As a matter of fact, such conditions are occasionally referred to him by the physician for consultation.

Although it is true that the periodontist is apt to get more such cases than is any other practitioner, certain conditions occur so rarely in his individual practice that he is deprived of sufficient opportunity to study them. It is logical then to believe that a pooling or an interchange of experiences and observations on unusual cases by many of our members should be advantageous and should result in much scientific enlightenment and clinical progress. Therefore, as a member of the Committee on Scientific Investigation, I wish to suggest that, if my expression meets with the approval of this Academy, special and ample time be hereafter set aside on our annual program for such a purpose. It is with this idea in mind that I present the following two interesting cases, each of which shows a marked acute hypertrophic and proliferative gingivitis, accompanied by a severe anemia.

Accumulated evidence collected from clinical data of cases of blood dyscrasias has established the fact that in many instances symptoms appeared in the mouth as the first sign of the disease. Oral manifestations of blood dyscrasias form a very important group of mouth lesions, and while I do not contend that it is possible to make a diagnosis of any form of blood dyscrasia by merely an oral examination, still one must feel that since the earliest symptoms of such disease appear in the mouth, early recognition would lead to early diagnosis and hence a more favorable prognosis.

It is with the thought in mind, that as dentists, we find ourselves confronted with the problem of putting the mouth in a healthy condition and of knowing something of the relation of general health to mouth health, that this paper is presented.

AGRANULOCYTOSIS

Primary granulocytopenia, agranulocytic angina, and polymorphonuclear leukopenia are descriptive terms meaning a marked increase in or total of granular leukocytes in the peripheral blood.

Appleton[3] was the first to attract the dental profession to this condition in a comprehensive report given before the New York State Dental Society in 1931. He chose as his subject "Agranulocytosis" because "of the widespread interest shown in it at the present time and because it will illustrate the essential unity of medicine, particularly the interrelations between oral conditions, on one hand, and extraoral or general conditions on the other".

INTRAORAL LESIONS

Lesions on the mucous membrane of the mouth may appear as a gingivitis or an extensive stomatitis. They have been described by various authors as "painful necrotic ulcers", "edema of the palate with ulceration", "large nonmarginated ulcers on both cheeks (orally) and in the floor of the mouth".

My interest in blood dyscrasias began in 1930, at which time I examined the mouth

*Read before the American Academy of Periodontology, July 31, 1947, *Boston, Massachusetts.*

of an acutely ill female, aged 45 years, who was a patient in the University Hospital (Philadelphia) and had been diagnosed as having "agranulocytosis". There was an ulcerative stomatitis present which the patient said began as a gingivitis seven days previous to admission.

During the period from March 8, 1930 to October 9, 1931, the patient had been admitted six times, four as an emergency for treatment and two for check-up. She spent a total of 114 days in the hospital and her mouth was treated each day with a variety of drugs and mouth washes.

The significant fact in this case was that as the disease was revealed in the blood study, it was also manifested in the mouth, as the blood count returned to approximate normal limits so did the condition of the gingival tissues and the mucous membrane of the mouth.

The patient was last seen four years after discharge. She was apparently in good health and her oral tissues were within normal health limits.

The second patient to be seen with the diagnosis of "agranulocytosis" was a dentist, aged 42 years; whose trouble began when the teeth became "abscessed" and were removed. The disease terminated fatally when the patient failed to respond to treatment.

Christian and Hinton[4], Flexner[5], Franken and Hensel[6], Leys[7], Aison[8] and others reported cases in which tooth removal was closely associated with the development of the disease.

Kracke[9], Fitz-Hugh[10], Watkins[11] and others felt that amidopyrine was a factor in the disease. For years pharmaceutical manufacturers and laboratories that manufacture dental preparations have made it a practice to send complimentary samples of secret proprietary mixtures containing amidopyrine to dentists. It is probably true that dentists have indiscriminately prescribed these preparations for postoperative pain following the extraction of teeth, often probably being guilty of giving the preparation to the patient in the original box in which it arrived, which no doubt has played some part in influencing the patient to use them for headaches and other physical discomforts. Because of the dangers from self-administration, a number of state health departments have limited the sale of a great many amidopyrine combinations to prescriptions from physicians and dentists.

In answer to the question, "Is amidopyrine primarily responsible for the production of granulocytopenia?" the Council of Pharmacy and Chemistry, of the American Medical Association replied, "As far as can be learned from evidence at hand, there can be no question that amidopyrine is very important in the production of granulocytopenia. In fact, no other agent has been found, either chemical or bacterial, which had been a factor in causing so many attacks."

Nixon[12], Madison and Squier[13], reported agranulocytosis which followed the administration of the sulfonamide compounds, this was also confirmed by Willius and Brown[14].

LEUKEMIA

According to Kracke and Garver[15], leukemia is a progressively fatal disease, characterized by a widespread hyperplasia of the hematopoietic tissues; resulting in the production of excessive numbers of immature white cells, which either circulate in the blood stream or become deposited in the fixed tissues, or both.

Following the elaboration of new methods of staining blood cells and the greater precision of differential counts of the blood, leukemia was divided into myeloid and lymphatic forms, and in both of these types there were acute and chronic stages. No form of treatment, even palliative, has been found for the acute forms of leukemia which run the course of a fulminating overwhelming infection with rapidly fatal outcome[16].

An elevated blood count is not always essential to the diagnosis of leukemia, approximately 45 per cent of the cases of acute leukemia seen at the Mayo Clinic had total leukocyte counts of 10,000 per cubic millimeter. It is the qualitative rather than the quantitative picture that is important. Morphologically the finding of large numbers of immature leukocytes with a high proportion of "stem cells" (myeloblasts) in stained films of blood establishes the diagnosis[17].

Whitby and Britton[18] describe two theories:

The Infective Theory. During a severe infection there is a large increase in circulating leukocytes. At the same time there is a marked shift to the left in the polynuclear count, with the appearance sometimes of a few myelocytes and occasionally myeloblasts.

This reaction sometimes suggests that there may be an infective cause for leukemia.

The Neoplastic Theory. The leukemic process resembles a malignant tumor. The manner of growth is the same and the leukemic cells infiltrate organ tissues to a certain extent and give rise to tumor-like growths. The disease is invariably fatal, and many cases show gradations between true malignant diseases and clear-cut leukemia. Against the neoplastic theory it may be said that leukemia starts in every place at once, and it is very rare to find any infiltration or penetration of the capsules of organs.

The disease was described by Bennett[19] over a hundred years ago, which he recognized as a new clinical entity characterized by "suppuration" in the blood accompanied by enlargement of the spleen. He called it leukemia.

Although much progress has been made in the recognition and classification of leukemia, there has been little improvement in the methods of treatment, since the life span of the patient with leukemia today is little more than it was one hundred years ago.

Distribution and Incidence:

Leukemia has wider distribution throughout the entire animal kingdom. It has been reported in avians and mammals, including chickens, mice, pigs, horses, cattle, birds and dogs. When it occurs spontaneously in these animals there is little to distinguish it from the disease that is seen in man, and apparently it is the same disease.

Rosenthal and Harris[20] believe that males are more frequently affected than females, the ratio being about three to one; and there seems to be no racial susceptibility.

Oral Lesions:

Maloney[21] feels that the oral lesions in acute leukemia are important clinically, not only because they occur in a large percentage of cases, but also because they are the earliest sign of the disease. The oral lesions are confined mainly to the gingival tissues and mucous membranes. The gums are swollen due to infiltration of the leukemic cells. At times the swelling is so extreme that the gums cover the teeth.

Cooke[22] reported that "stomatitis occurs with sufficient frequency to be considered one of the symptoms of acute leukemia". He stated, however, "that it is not common in children and was only noted in eight of our cases out of fifty-five". The usual type is an ulcerative stomatitis with some swelling, redness, and purulent exudate at the gingival margins.

Severe stomatitis with bleeding as well as ulceration of the gums and fauces; a diagnosis of diphtheria is sometimes made, and not uncommonly the dentist may have removed a tooth on account of sepsis. It is possible that the oral ulceration is due to the absence of polymorphonuclear cells from the blood, in which case the lesions are produced in the same way as in agranulocytic angina. There is a generalized hemorrhagic tendency, especially in the terminal phases, which is probably due to a diminished number of platelets in the blood. Extensive purpura with oozing of blood from the gums and mucous membranes may be found. The lymph glands are not enlarged as a general rule, but the cervical group may be involved as the result of the infective condition in the mouth.

CASE REPORT NO. 1

A white male, aged 52, applied for treatment at the Dental School, University of Pennsylvania on October 12, 1940. He was referred by his family physician.

Previous History: A month previous the patient felt somewhat unwell, he fatigued easily, did not feel like eating because his "gums were sore". He visited his family physician who prescribed a tonic.

Two weeks later he noticed that "the infection was spreading in his gums and that his breath was awful". His physician referred him to a dentist, who made roentgenograms of the teeth and whose report was as follows:

6 October 1940

"Dear Doctor.....,

"Enclosed find X-rays for your patient Mr. ...., which show as follows:

"1. The upper right bicuspid shows chronic apical infection involving the surrounding bone infection.

"2. In the upper right cuspid region there is an impacted cuspid with a horizontal inclination.

"3. The upper left cuspid shows root remaining.

"4. The upper left second bicuspid shows periapical infection.

"5. The upper left first molar has absorption of bone at bifurcation of roots.

"The upper and lower teeth have absorption of

bone around roots, especially the lower anteriors.

"Would advise the removal of infection.

"Trusting this information is satisfactory and thanking you, I am.

Sincerely yours,

....., D.D.S."

The patient was then referred to the Dental School Clinic.

The patient was of average nutrition and development. He was an intelligent and most coöperative patient, who did not seek charity but came to the Clinic because his physician referred him for the removal of teeth.

Findings: There was a marked gingival hypertrophy present about the remaining teeth and ulcerated areas which bled easily. The gums were not painful, but they were slightly discolored and there was marked fetor oris.

Fig. 1, Case 1. Photograph taken when patient first applied for treatment shows marked gingival hypertrophy with areas of ulceration which bled easily.

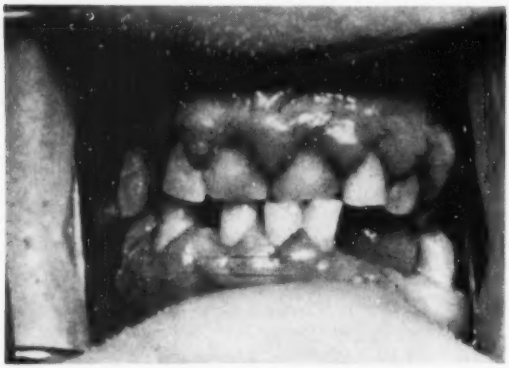


Fig. 2, Case 1. The general condition of the gingival tissues improved while patient was receiving blood transfusions and supportive measures while in the hospital as well as daily care of the mouth.

Treatment: Because of the general appearance of the gums, the patient was instructed in oral hygiene and taken to the Haematological Department of the University Hospital for complete blood studies and physical examination. This examination revealed acute leukemia, which warranted admission to the hospital immediately.

The blood count was as follows: R.B.C. 2,800,000, Hgb 46 per cent, W.B.C. 205,000, Polys. 12, Lymph 2, Monos. 0, Eos. 1, Myel. 16, Platelets 14,500.

Course of Disease: On admission the patient was placed on zinc peroxide mouth wash and the gums were treated daily.

Medical treatment included transfusions and supportive measures and at the end of a month the patient felt better, had gained several pounds, and was anxious to return home. Along with his general improvement, the condition of the gingival tissues was better, the bleeding had ceased, and the ulcerated areas appeared to have responded to the general medical treatment. He was permitted to return to his home after a month in the hospital and was followed weekly as an outpatient.

In less than a month the patient became acutely ill and was readmitted, there was marked gingival hypertrophy with areas of necrosis throughout the mouth. The gums bled freely and it became difficult to give relief even for the marked fetor oris.

The course of the disease continued in spite of medical treatment, and as the disease progressed, the general appearance of the gums became worse until the hypertrophied gums extended to the incisal edges of the teeth and there was a generalized necrosis.

At autopsy the gums exhibited a re-

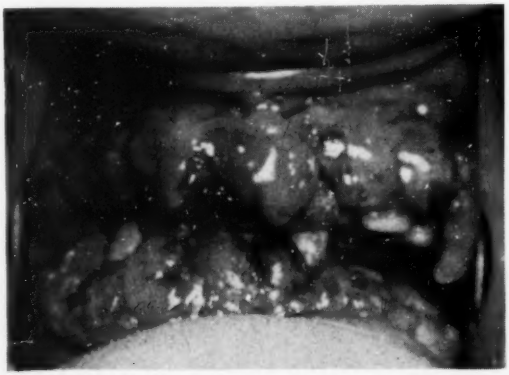


Fig. 3, Case 1. After a short interval of improvement the course of the disease went on to fatal termination. The general hypertrophy of the gums with necrosis continued with the progress of the disease until the teeth were practically obscured by the gums.

markable grayish-green and hemorrhagic, fleshy, friable thickening which extended down over and completely covered the teeth.

Histologic diagnosis "Leukemia infiltration, marked".

CASE REPORT NO. 2

A white male, aged 46, came to the Oral Diagnosis Clinic, University of Pennsylvania Dental School, on March 26, 1938. His chief complaint was "sore gums", that they felt "ropey" and he was unable to chew his food because the gum "was over the teeth" in the molar region (Fig. 4).

Previous History: The patient who is a tailor, was well until three weeks ago, when after drinking a glass of water he felt a burning sensation in his throat. Two days later he had difficulty in swallowing and the soreness now included his tongue, gums, and teeth. He visited his family physician who prescribed a mouth wash and capsules for a sore throat. The patient received no relief and told his story to a dental student who was in his shop and who brought him to the school.

Fig. 4, Case 2, There was a marked gingival hypertrophy, in the molar region it reached the occlusal surface of the teeth. There were areas of necrosis throughout and marked fetor oris.

Findings: There was a marked gingival hypertrophy throughout, in the molar region the gum was up to the occlusal surface of the teeth. There were areas of ulceration and necrosis throughout, the gums bled easily and there was marked fetor oris.

The submaxillary, anterior and posterior cervical nodes were enlarged and freely movable.

The tonsils were markedly enlarged and the pharynx reddened.

The patient was taken to the Medical outpatient department for examination and was found to be suffering from acute leukemia. He was admitted for study and treatment to the University Hospitial.

Repeated blood studies and sternal puncture confirmed the diagnosis of acute leukemia and in spite of blood transfusions and supportive medical care, the patient failed to respond to treatment.

Before fatal termination of the disease dental radiographs were made, which on comparison with the original films disclosed a marked destruction of alveolar process in both the maxilla and mandible (Fig. 5). Necrotic ulceration of the gums extended to the palate and posterior beneath the tongue to the molar region.

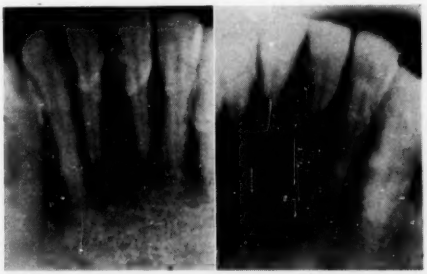
MONOCYTIC LEUKEMIA

According to Sturgis[23] "within recent years it has been recognized that a leukemia may occur which differs from other types in that there is a significant increase in the cells of the monocyte series in the circulating blood. The characteristic pathologic change is an infiltration of the organs of the body, especially the spleen and the liver, with monocytic cells resembling those of the blood".

Distinctive clinical features appear to be the frequent occurrence of oral lesions such as gingivitis and especially necrotizing, ulcerative gingivitis, and a tendency for the patients to develop purpura and bleeding from the mucous membranes.

Osgood[24] believed in 1937, that monocytic leukemia was more common than had previously been thought. He stated that swelling of the gums was present in 80 per cent of the cases and was often associated with gangrenous stomatitis.

Watkins and Hall[25] found that in three of the nine acute cases and in two of the fourteen chronic cases of the Naegeli type of monocytic leukemia there was swelling and bleeding of the gums. None of the patients who had the Schilling type of monocytic leukemia had involvement of the gums. In certain instances, swelling and bleeding of the gums were followed by ulceration and



a b

Fig. 5, Case 2. (a) Roentgenogram of mandibular incisor teeth made when patient was first seen. (b) Roentgenogram of same areas as (a) which was made six weeks later, at the termination of the disease showing alveolar bone destruction.

necrosis of the gums and buccal mucous membrane.

Montgomery and Watkins[26] reported a case in which "Examination revealed hypertrophy of the gums, mucous membranes, and soft palate, which was swollen and edematous. The lymph nodes in the neck were enlarged, tender, discrete and movable".

Forkner[27,28], Dameshek[29], and Mitchell[30] have emphasized infiltrative lesions of the gums as more often associated with acute monocytic leukemia than with other forms of leukemia.

Fig. 6, Case 3. A marked gingivitis was present especially about the mandibular incisor teeth, it appeared as one of the first signs of the disease (leukemia) and was the patient's chief complaint.

CASE REPORT NO. 3

An acutely ill, pallid white girl was seen at the University Hospital in Philadelphia in October, 1938.

In part, the physical examination revealed palpable lymph nodes, varying in size from that of a pea to several the size of marbles, on the submaxillary anterior cervical, axillary and epitrochlear chains, which were discrete, firm, and non-tender.

Examination of the mouth showed the teeth to be in excellent condition. There was a marked gingivitis present, especially about the mandibular incisor teeth with some hypertrophy. The soft palate and oropharynx were acutely inflamed and there was a marked fetor oris.

A study of the patient's blood and marrow puncture of the sternum established a diagnosis of acute monocytic leukemia.

On the fifth day after admission the submaxillary glands were considerably swollen and the mouth condition was the chief complaint. The mucous membranes were painful, ulceration extending posterior in the lower jaw to the molar region. On the lingual surface the gums were swollen and there was some ulceration. There was a marked fetor oris. The patient's family insisted on taking her home where she died in 48 hours.

Summary: In this case symptoms appeared in the mouth as one of the first signs of the disease and the patient's chief complaint was swollen and painful gums with the marked fetor oris.

CONCLUSION

It is a known fact that the first signs and symptoms of blood dyscrasias may make their appearance on the mucous membrane of the oral cavity. It would therefore be unwise to attempt any treatment in suspicious cases without first seeking consultation and reaching a definite diagnosis.

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Some Aspects of Inflammation*

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IT IS INDEED a great pleasure to address the American Academy of Periodontology.

It would be time-consuming and probably confusing if I were to take up the numerous aspects of the interesting phenomenon of inflammation which has been especially studied for the last twenty years. I will, therefore, discuss with you only those phases which may be of special interest to you.

Inflammation is the physical basis of infectious processes in the vertebrate kingdom. Nevertheless, one should not infer that infection and inflammation are synonymous terms. Inflammation is essentially a manifestation of severe cellular injury in the higher animal forms; whereas infection is one particular type of cell injury caused by a viable agent. Therefore the term "inflammation" is quite inclusive, while the term "infection" is definitely more restrictive in its meaning. An inflammation may appear merely as a hyperaemic reaction. It may progress and become a true suppuration[1]. It is difficult for me to classify these various manifestations as caused by essentially different mechanisms. It is of little moment whether one studies inflammation in the periodontal tissue or in some other part of the body. The principles involved are exactly the same and it is some of these principles which I would like to consider at this time.

It is interesting to note what happens in the initial stage when any sort of irritant is introduced into an otherwise normal tissue. There is at first a disturbance in fluid exchange. This is referable to a transitory increase in capillary pressure; but of greater importance is the enhancement in capillary permeability which accompanies any form of inflammation. This lasts throughout a large period of the inflammatory reaction, and it is probably the most important of initial changes in the development of inflammation. Inasmuch as all later sequences in the so-called inflammatory reaction depend upon this pivotal reaction of increased capillary permeability, it would be well for us to discuss the mechanism involved.

When a dye is injected into the ear vein of a normal rabbit, it takes hours before the animal becomes diffusely blue. On the other hand, if an animal has an inflammation in several tissue areas, the dye (trypan blue) penetrates rapidly into these inflamed areas owing to the local increase in capillary permeability. The same can be shown with bacteria or graphite material. It is thus clear that the extent of increased capillary permeability is quite appreciable with inflammation. What is the mechanism concerned? This is obviously an important question if one is to fully understand any of the sequences involved in the development of the inflammatory reaction[2,3].

When exudative material is introduced into the skin of the abdomen of a rabbit, and when this is followed by the intravascular injection of a dye, the dye rapidly accumulates in the skin area originally treated with the exudate. This has suggested that in the exudate a permeability factor is liberated by injured cells, for this cannot be obtained when instead of exudative material blood serum is injected into the skin. This permeability factor has been extracted by a rather laborious technique after removal of the protein fractions. The final material is highly active in inducing increased capillary permeability. This material not only causes greater permeability response on the part of capillaries, but it also causes the migration of leukocytes to the site of injury[4]. This material has been called, as you well know, *leukotaxine*. It was isolated in the laboratories of the Harvard Medical School some ten years ago. By utilizing a somewhat different technique, Cullumbine and Rydon (in England) have confirmed the presence of leukotaxine in exudates[5]. Leukotaxine seems to be a polypeptide to which there can be attached a prosthetic group, the nature of which is not yet known.

*Presented before the American Academy of Periodontology, Aug. 2, 1947, *Boston, Massachusetts*

I regret that time does not permit me to go into further details of our various studies on leukotaxine which, incidentally, has no relation to histamine. Nevertheless, I hope that I have pointed out clearly to you the importance that this substance has in explaining reasonably well two of the fundamental mechanisms involved in the development of inflammation, namely, the initial increase in capillary permeability and the migration of polymorphonuclear leukocytes to the site of an acute injury.

There is another substance liberated in inflammatory exudates which I should like to mention, briefly, for such a substance may be of definite interest to you. Leukotaxine as such induces very little injury to normal tissues besides increasing capillary permeability and causing the migration of polymorphonuclear leukocytes to the site of an acute inflammation. Yet injury with inflammation is a fairly well stereotyped reaction. The earlier writers, such as Celsus in the middle ages, recognized that there are four cardinal features in inflammation. You are well acquainted with these. To the "rubor, callor, tumor, and dolor" of Celsus and the "loss of function" of John Hunter, we may add another biochemical cardinal feature, namely, proteolysis. It is true that an irritant possesses its individual chemistry and that this together with the anatomical location of the area of injury may eventually modify the picture of the inflammatory lesion. Nevertheless, close scrutiny indicates that the pattern of injury in all sorts of inflammation is quite constant. The parenchymatous organs may reveal merely cloudy swelling, but in general the cardinal features of Celsus are the primary signs of an inflammatory reaction. The problem which faced us centered around the mechanism of injury in inflammation. In brief, it was shown that in the euglobulin fraction of exudates there is present a toxic substance evidently liberated by injured cells[6]. This toxic substance contains proteolytic activity[7]. It has been termed "necrosin". The indication to date strongly suggests that we are dealing with a true euglobulin, but it is still quite possible that further studies will reveal that necrosin is merely associated with the euglobulin fraction of exudates. The introduction of this substance into tissue is followed several hours later by an acute erythema, swelling, and central necrosis. The lymphatic vessels are occluded. In earlier studies, we have shown this to be a gauge of the extent of cellular injury. Such tissue injury fails to occur when one injects the euglobulin fraction of blood serum. Several minutes after the injection of necrosin into the cutaneous tissue of a rabbit there is swelling of the collagenous bundles[6]. This fails to occur following the introduction of the euglobulin of blood serum. Necrosin can at times be recovered from the blood serum of an animal with a concomitant acute inflammation. This suggests that the toxic substance liberated by the injured cells can occasionally penetrate into the systemic circulation. This fact is of definite importance when one deals with what apparently is merely referred to as a "local acute inflammation". The presence of necrosin and its recovery from the blood offers a reasonable explanation of the effect of foci of infection on organs located at a distance. In fact, when necrosin is injected into the blood stream of an animal, there is usually some damage to the liver and frequently, though not as consistently, to the kidney. Other structures may or may not show some degree of damage. Repeated intravascular injections of necrosin into an animal, in an endeavor to duplicate the continuous liberation of this toxic material from a focus of long-standing inflammation, results in damage to the liver. The hepatic cells appear markedly vacuolated. This appearance is referable to either an abundance of fat or glycogen deposition in the cytoplasm of these cells[8]. The liberation of such a toxic substance by injured cells doubtless is of significance to the student of inflammation, irrespective of the site of injury.

There are two other components in the euglobulin fraction of exudates which, owing to the limit on time, I can but barely touch upon. There is a substance, that can be dissociated from necrosin, capable of inducing fever. This substance provides a reasonable explanation for the mechanism of fever with inflammation[9]. It has been termed "pyrexin". Another component of the euglobulin fraction of exudates, is a factor causing a decrease in the number of circulating white cells. This leukopenic factor is closely associated with pyrexin[10]. It is this leukopenic factor together with another opposing factor present in the pseudoglobulin fraction of exudates which determines the ultimate level of white blood cells in the circulating blood. The other factor in the

pseudoglobulin fraction of exudates is known as the "leukocytosis-promoting factor" (abbreviated as the LPF). It offers a reasonable explanation for the mechanism of leukocytosis with a large number of inflammatory processes[11,8]. It is unfortunate that time prevents me from going into a detailed discussion of the various substances liberated by injured cells into inflammatory exudates. Nevertheless, I believe that by stressing even two of these substances you can well see the significance of enlarging our studies on the biochemistry of injured cells.

Finally, I would like to discuss with you certain other features which point out the necessity of understanding the various chemical reactions which occur in the development of inflammation. I would like to stress the changing hydrogen ion concentration as the inflammatory reaction progresses, as well as to point out the mechanism of local acidosis involved[12,13]. The morphological implications of these chemical changes will become obvious. In the first stage of inflammation the pH of the exudate is alkaline. It may range between 7.2 and 7.4. At this stage the prevailing cell is the polymorphonuclear leukocyte. With the progress of the acute inflammatory reaction or by intensifying it by merely reinjecting the irritant, the hydrogen ion concentration rises. A state of local acidosis occurs. The polymorphonuclear leukocytes are unable to survive. They become replaced by macrophages. The shift in pH can explain adequately the histological shift from polymorphonuclears to macrophages, well known to occur in inflammation. The pH at the stage of macrophages ranges between 6.8 and 6.9. As the pH sinks to even lower values, that is, below 6.5 or so, all cells are affected and we have a state of suppuration. Thus the production of pus is essentially a function of the hydrogen ion concentration.

The basic question, however, lies in the determination as to what constitutes the mechanism of local acidosis with acute inflammation. It is true that the alkali reserve is reduced in the inflamed area, but this is essentially a restatement that there is a local acidosis. The acidity in the circulating blood stream is unaltered, probably due to the effective buffer mechanism. Cells in general have a double potentiality of obtaining their oxygen supply. Oxidation ordinarily is performed through the contact of oxygen carried by red blood corpuscles to the tissues. If, however, the local circulation is damaged or inadequate, the energy required is derived by the process of anaerobic glycolysis. This implies the conversion of glucose to lactic acid. In an acutely inflamed area, with the blockade of lymphatic structures which is caused to a large extent by the liberation of necrosin, the local circulation is markedly impaired. With progress in intensity of the acute inflammatory reaction the vascular channels are likewise damaged, and the area of injury is shunted, so to speak, from the rest of the organism. Under such circumstances the remaining and relatively viable tissues at the site of injury derive their energy from anaerobic glycolysis. A considerable amount of lactic acid is formed. The result is a true local lactic acid acidosis. This accounts for the rise in hydrogen ion concentration, with the corresponding cytological changes described above. In brief, it is clear that the cellular content at the site of an acute inflammation is regulated by the pH, which in turn is referable to a disturbance in intermediary carbohydrate metabolism. There are other changes in carbohydrate metabolism at the site of an acute inflammation. For instance, injured cells are capable of producing glucose and this explains to a large extent, in the absence of insulin in the diabetic organism, the enhanced state of the blood sugar level in this disease, whenever there is a concomitant inflammation[14,15]. Necrosin is also capable of liberating a certain amount of glucose, perhaps from the liver into the circulation[7]. This fact is also of significance in diabetes. In this disease, when there is an accompanying inflammation, glucose is formed locally by injured cells, by deamination of the protein molecule, while a certain amount of sugar is possibly liberated from the liver by the action of necrosin. The combination of these two processes tends to enhance the blood sugar level whenever diabetes is complicated by an inflammation.

Necrosin, pyrexin, and the leukopenic factor are usually formed in greater abundance in acid exudates. Consequently, as the acute inflammation progresses in intensity more lactic acid is formed locally, the hydrogen ion concentration rises, and the production of necrosin is appreciably increased. Necrosin induces lymphatic blockade which in turn tends to localize the area of acute inflammation.

CONCLUSION

I believe I have sufficiently stressed the close interrelationship between the local formation of toxic material, such as necrosin, at the site of an acute inflammation, and the implication of this substance in regard to impaired lymphatic drainage. I have also emphasized the significance of the local hydrogen ion concentration on the morphology of the prevailing cells at a given time in an area of acute inflammation.

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Nutrition and Dental Health

HUMAN FITNESS AND THE STATE OF VITAMIN NUTRITION

Condensed from: Keyes, Ancel; *Nutrition Reviews*, 5:129. May, 1947.

By DOROTHEA F. RADUSCH, D.D.S.

GOOD NUTRITION has been defined in many ways. The challenging nature of the concept appears in the fact that many of the definitions are open to criticism on practical or theoretical grounds. One definition, which perhaps may be defended as well as any, is that good nutrition is that condition which permits the development and maintenance of the highest state of fitness. The onus then is shifted to the term "fitness" which is troublesome to define precisely because, to judge from its popularity in usage, everyone knows what it means, but not how to measure it. Broadly, fitness embraces occupational work capacity, ability in physical and mental tasks, and power to withstand physical and biologic stresses. Even if there is arbitrary restriction to a particular aspect, such as "physical fitness", its evaluation is a complex and difficult problem.

Fitness connotes positive health as distinguished from disease so we are not here primarily concerned with the states of marked unfitness which occur in

classic vitamin deficiencies. It is pertinent to examine the effects on fitness of milder deficiencies. Conversely, there is the possible effect of unusually abundant vitamin intakes. Finally, there are the questions of the time course of alterations in fitness after changing the vitamin intake.

A major point at issue is whether fitness is peculiarly sensitive to differences in vitamin nutrition, so that changes in fitness precede the development of other effects.

It is essential to recognize the limitations of current knowledge. Except in unusually well controlled experiments, not much reliance can be placed on reports of the subjects themselves or the subjective impressions of observers. The number of acceptable investigations on vitamin-fitness relations is small and they have dealt with relatively short periods of time—from a few days to about a year—in the life of persons representing only a small part of the population; the great majority of cases have been healthy males between 18 and 30 years of age.

With these rather severe limitations in mind, the great bulk of evidence indicates no gain in fitness of adults from vitamin supplementation added to a good American diet. There are opposing claims in the literature but most of these have so little basis as to deserve no separate mention.

The few experiments conducted so far have given no support to claims that supplementary vitamins increase fitness in hot weather. Much the same seems to be the case in cold environments although only one major experiment has been reported.

Evidence is gradually accumulating regarding the time course of events following a change from a fairly good diet to one definitely low in vitamins. Changes in physical fitness are neither early nor especially prominent results of diets extremely deficient in vitamin A. Subsistence on a diet very low in ascorbic acid eventually produces a decline in physical fitness but this does not seem to precede the more characteristic changes in the gums, capillary resistance, and wound healing. With both vitamins A and ascorbic acid it is not proper, from the present evidence, to suggest that low states of fitness may be ascribed, in the absence of other signs, to deficiencies in these vitamins.

The greatest interest regarding fitness has centered on the vitamins of the B-complex. On diets in which the major B-vitamins are reduced to 34 to 40 per cent of the National Research Council's Recommended Daily Allowances, elaborate and well-controlled studies have failed to disclose any loss in physical or mental capacity within periods up to at least six months. When these vitamins are reduced to extremely low levels in the diet, the first notable effects, which may appear in a week or two, are anorexia and mental depression; some of the subsequent loss of fitness may be due to plain starvation. After the effects from general deprivation of B-vitamins are well established, the provision of thiamine alone tends to correct the situation, indicating that under these conditions thiamine is more critical than the other B-vitamins.

The several aspects of fitness are not equally responsive to acute restriction of the B-vitamins. It appears that simple strength, intellective capacity, and the special senses are remarkably resistant, that endurance is much more sensitive,

and that psychomotor functions like neuromuscular coördination and speed occupy an intermediate position. The most important point is that in all acceptable experiments so far reported, changes in fitness have been preceded by one or more of the following: anorexia, personality changes, signs of polyneuritis, oral or cutaneous alterations, increased pyruvic acid in the blood.

The general conclusion from present evidence is that vitamin supercharging confers no benefit in terms of fitness and that fitness is not peculiarly sensitive to vitamin deficiencies. Better methods for evaluating fitness are needed, and it must be pointed out again that this term does not include many other aspects of health such as maternal and infant health, longevity, and disease resistance, in which clearly demonstrable relations to improved nutrition have been shown.

Comment: There are two considerations to bear in mind regarding this discussion; the first, the restricted meaning of the term "fitness" as used by Keys. Surely more common usage gives to it a broader meaning of over-all good health, including then, phases omitted by Keys, in particular, mental states and personality changes. This points to the importance of defining terms used, as was done by Keys.

The second consideration is that the conclusions regarding lack of benefit from supplementation apply only when vitamins are added to a *good* American diet. There is a question as to how many diets of patients would fall into the category of "good diets".—D.F.R.

RELATION OF INHERITANCE TO NUTRITIONAL REQUIREMENT

Condensed from: *Nutrition Reviews*, 5:237. Aug. 1947.

THE DISTINCTION is made often, that the determination of a biologic event is due to (a) environment, or (b) heredity, or as Francis Galton once put it "to Nurture or to Nature". The utility of a science of nutrition is predicated on the base that the nutrition of an organism is part of its environment and hence an understanding of the facts of nutrition leads facilely to a control of biologic events in which the organism participates. There is recognition that certain biologic events are completely under the control of heredity, while environment, including nutrition, plays no part in their determination, e.g., the blood groups.

Even those items of the nutritional environment which are effective in their influence on certain biologic events must operate in a genotype, and if genotypes vary, as they certainly do, then it must be anticipated that at some time the growing science of nutrition must come to grips with the problem of genotype. This is well recognized right now at the species level and is recognition of the fundamental reason why, for example, ascorbic acid as an item of diet has greater meaning for guinea pigs, monkeys, and man than it does for the rat or the mouse. At the intraspecific level, information is meager. As the science of nutrition expands we may expect increasing attention to the limits that the genetic composition of an organism puts on the effect of nutrition, or

put another way, to the diversity of the effects of a nutrient as it operates in diverse genetic circumstances.

Some beginnings have been made, primarily in animal experimentation. Significantly different efficiency indexes of food utilization have been shown between the two sexes in rats, while different rates of utilization were established for two lines of rats, the lower being about 40 per cent less efficient than the high efficient line. Strikingly different requirements for thiamine and for choline in strains of rats have been reported, as well as variations in life expectancy, not due to diet. Chicks have been developed which are relatively resistant and others relatively susceptible to riboflavin deficiency. Differences in hatchability of eggs have been observed, which are presumed to be genetic in origin.

Other variations of varying responses to the same diet by different representatives of the same species have been described. In dental caries investigations, different strains of white rats have been observed to have different susceptibilities to the development of carious lesions. The development of strains of widely different caries resistance and susceptibilities have been produced by selective inbreeding. Hamsters and cotton rats too show these strain differences.

These varied examples of relations of inheritance to nutritional requirements demonstrate clearly the difficulties which may arise in the evaluation of the requirements of various nutrients for a given species. The necessity for making allowances for individual variations in a human population becomes more obvious when such data are considered.

In Memoriam

JOHN V. COGAN

1890-Sept. 10, 1947

Editorial

Salt and Soda

THE use of sodium chloride for cleaning the teeth has been recommended to patients by the occasional dentist for many years. Sodium bicarbonate has also been recommended. In more recent years, this form of dentifrice (a combination of the two drugs) is frequently advised, especially by public health workers as economical, convenient, and effectual. One is surprised to learn how frequently this same advice is given by dentists to their patients. Fortunately, the unpleasant taste has prevented many from following this advice. At one time, a combination of equal parts of sodium chloride, sodium bicarbonate, and sodium perborate was recommended by many dentists. Later, the Council on Dental Therapeutics of the American Dental Association published the following statement: "The Council . . . will not accept dentifrices for daily use which contain sodium perborate because of the mouth lesions and edema of the lips sometimes resulting from its use." This Council states also in *Accepted Dental Remedies* that, "Dentifrices containing frank medicinal agents will not be accepted." Apparently the Council does not consider sodium chloride and sodium bicarbonate "frank medicinal agents", because of its recent statement that, "Cleansing dentifrices are essentially scouring agents, and this function can be performed quite satisfactorily for most people with a mixture of finely powdered salt and baking soda." (*J.A.D.A.* 35(7):522. 1947.)

Sodium chloride is an emetic, a laxative, and has mild irritant properties. Sodium bicarbonate is used to neutralize acids and is frequently used in self-medication for various digestive disturbances, a practice disapproved by physicians.

May not a limited amount of these drugs be regularly ingested by those who use them to clean their teeth one or more times daily?

Most dentists are today mouth cancer conscious and are trying to avoid all forms of oral tissue irritation. Therefore, would it not be more consistent to recommend a dentifrice, if any, made up almost entirely of substances lacking pronounced chemical action on the soft tissues of the mouth. The effect of the salt and soda combination used in a healthy mouth can be felt for an hour or more. It may be from the dehydration of the tissues by the sodium chloride. If action from either of these drugs is called for, would it not be preferable to use them in solution or in very limited amounts with other cleaning agents? Finely milled sodium chloride is recommended, but one can safely assume that the average patient will use from the always available supply in the kitchen, and generally from that source the abrasiveness of the sodium chloride crystals are something to reckon with.

This Editorial is not to be interpreted as a criticism of the Council on Dental Therapeutics whose contribution to dentistry over the years has been one of continuous constructive service which all too often has been a thankless job. It has done more than any other single agency to make dentists conscious of their responsibility when recommending drugs. —G.R.S.

Nomenclature In Periodontia

(REPORT OF THE COMMITTEE ON NOMENCLATURE OF THE AMERICAN ACADEMY OF PERIODONTOLOGY—1947)*

Due to the efforts of the previous Nomenclature Committee, considerable clarification in the use of terms in periodontia has been reached. There are relatively few terms which have to be discussed or changed at this time. In this report we will repeat the accepted terms as they were published in the July, 1943 issue of the *J.A.D.A.* (p. 1112) and will include certain modifications and explanations as were suggested by the present committee.

1. *Alveolar bone.* The alveolar processes of the maxillae and the mandible. Within the alveolar bone, a distinction should be made between the "alveolar bone proper" and the "alveolar supporting bone" due to their biologic and functional differences. The alveolar bone proper may be defined as that thin perforated bony plate surrounding the root to which the fibers of the periodontal membrane are attached and through which the vessels and nerves enter and leave the periodontal membrane. In radiography this bony plate is called "lamina dura" as it appears more radiopaque than the surrounding spongy bone. The alveolar supporting bone consists of the compact alveolar plates and of the spongy or trabecular bone of the alveolar processes, other than the alveolar bone proper.

2. *Alveolus, dental.* The socket in the alveolar process in which the root of a tooth is held by the fibers of the periodontal membrane.

3. *Bruxism.* Gnashing or grinding of the teeth in other than normal function. This grinding of the teeth is an unconscious habit and is often limited to the sleeping period, but may occur at times of mental or physical concentration or strain. Bruxism may develop into a neurotic symptom, "bruxomania".

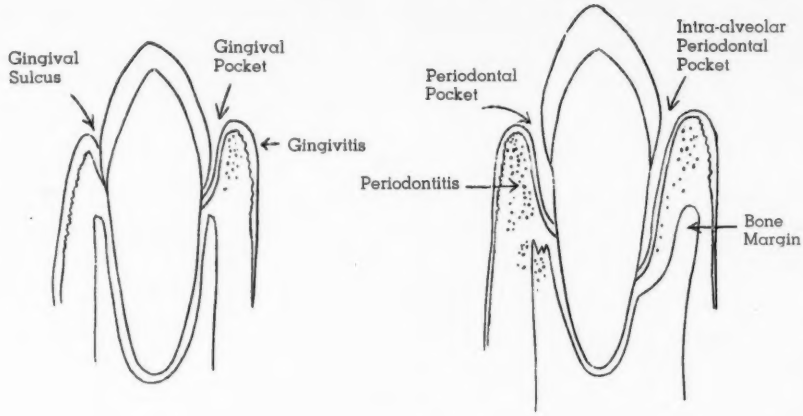
4. *Calculus. See—Dental Calculus.*

5. *Dental Calculus* (synonym—*tartar*). Calcareous deposit on the teeth (hard incrustation on the teeth, consisting of organic and mineral matter). Much confusion has existed due to the distinction between salivary and so-called serumal calculus, one said to originate from the saliva, the other from serum of the blood. This differentiation, however, is untenable and we can only distinguish between *supra-gingival* and *subgingival* calculus. The differences in consistency and color are due to the fact that in the pocket the deposits are more densely packed and are darkly stained from seepage of blood.

6. *Epulis.* The term "epulis", denoting a tumor on the gingiva, is a general term. The word originates from the Greek epi (on) ulon (gingiva) and means growth on the gingiva. It is a tumor of varying cell types, at times fibrous, at other times of giant cell character, vascular or inflammatory, etc. A proper differential diagnosis is only possible by biopsy.

7. *Gingiva.* That part of the oral mucous membrane that covers the alveolar processes of the jaws and surrounds the necks of the teeth. There has been considerable discussion about the proper pronunciation of this term (generally pronounced jin'jivah). All dictionaries (with the exception of Ottofy's *Standard Dental Dictionary,* 1923) give the pronunciation jin-ji'vah. There is no question that this would be correct if the rules of the Latin language were followed. However, it is recommended that the generally used pronunciation jin'jivah be maintained. This seems to be justified, first from common usage, second by the fact that the Latin word jin-ji'vah is no longer used as such but has become an English word. It is combined with English adjectives and not with Latin adjectives: i.e. buccal gingiva, palatine gingiva and not gingiva buccalis, gingiva palatina, etc. English adjectives can be used only with anglicized nouns: i.e. brachial artery, ulnar nerve, temporal bone, instead of arteria brachialis, nervus ulnaris or os temporale. The word jin'jivah can therefore be considered the anglicized form of the Latin jin-ji'vah. (Synonym: *gum.*

*Report *reprinted* from *J. Periodont.* 18(3):112-114, July 1947, because of corrections later made in the Advance Report. Presented and accepted at the Boston meeting of the American Academy of Periodontology, July 31, 1947.



The term "gum" should be eliminated from usage, especially in scientific literature.)

8. *Gingival hyperplasia*: Overgrowth of the gingival tissues. There is some misunderstanding as to the correct use of the terms hyperplasia and hypertrophy. A sharp and definite difference does not always exist. However, hyperplasia is defined as an overgrowth of tissue due to increase in the number of its elements; this overgrowth does not serve a useful purpose. Hypertrophy, on the other hand, is an increase of an organ due to increase of the *size* of its elements, serving a useful purpose. An example of a true hypertrophy is the enlargement of the heart due to overwork, or enlargement of muscles due to increased activity. Each cell becomes longer and thicker. Overgrowth of the gingiva, however, is an hyperplasia (increased number of cells).

9. *Gingival pocket.* Pathologic sulcus, deeper than the normal sulcus; the gingiva is in a pathologic inflamed condition (gingivitis). Its advanced stages are not sharply differentiated from the early stages of a periodontal pocket. (*See* diagram.)

10. *Gingival recession.* Progressive exposure of the root surface by apical shift of the gingiva, following resorption of the alveolar crest and loss of the corresponding part of the periodontal membrane. (*See—periodontal atrophy.*)

11. *Gingival sulcus.* The shallow groove around a tooth, bounded on one side by the surface of the tooth and on the other by the epithelium lining the free margin of the gingiva. This term designates the "normal" condition as distinguished from the gingival and periodontal *pocket.* (*See* diagram.)

12. *Gingivitis.* Inflammation of the gingiva. The condition may be further defined and described by such adjectives as acute, chronic, purulent, ulcerative, hyperplastic, scorbutic, etc.

13. *Intra-alveolar periodontal pocket.* This term designates the condition when the bottom of the pocket is beyond the original level of the crest of the alveolar process. (*See* diagram.)

14. *Oral Mucosa* (or *oral mucous membrane*).

Masticatory mucosa. Epithelium hornified, immovably attached

Gingiva

Hard palate

Lining mucosa. Epithelium non-hornified

Loosely attached

Firmly attached

Specialized mucosa. Back of tongue. (*See* Orban, B. and Sicher, H.: "Oral Mucosa" *J. Dent. Educ.* 10:94-103, 163-164, 1946.)

This classification of the different regions of the oral mucous membrane is necessary because of the confusion existing in naming the different parts. It is of special importance to differentiate between the gingiva (hornified, firmly attached) and the lining mucosa covering the alveolar process (non-hornified, loosely attached); these two different tissues are separated from each other by a festooned line.

It was questioned whether the term

"hornified" or "cornified" should be used. The terms "hornified" and "hornification" should be preferred as the English stem "horn" is in combination with an anglicized noun or participle.

15. *Periodontal abscess.* Acute purulent exacerbation of a chronic periodontitis, associated with deep periodontal pockets.

16. *Periodontal atrophy.* Progressive resorption of alveolar crest with loss of the corresponding part of the periodontal membrane and gingival recession.

17. *Periodontal diseases.* Collective term designating all periodontal disturbances.

18. *Periodontal membrane.* The fibrous tissue that connects the cementum of the tooth to the surrounding structures. "Pericementum" was suggested as a term to replace periodontal membrane by some members of the committee and consultants. However, while it is realized that the term "periodontal membrane" is not entirely correct the term "pericementum" does not eliminate its shortcomings. The main point is that the periodontal membrane is not a "membrane" but functions as a combined pericementum, periosteum, and alveolo-dental ligament. Since there is no adequate term to integrate these functions the well-known and recognized term of "periodontal membrane" is preferred. If analyzed, this term has less misleading implications than the term "pericementum". The term "pericementum" should be discarded.

19. *Periodontal pocket.* Pathologically deepened sulcus extending into the former periodontal membrane apically to the original level of the resorbed alveolar crest. The early stages are not sharply differentiated from the advanced stages of a gingival pocket. (*See* diagram.)

20. *Periodontal traumatism.* Injury of periodontium due to excessive occlusal operative, accidental or orthodontic stress. (*See —traumatism.*)

21. *Periodontia.* The branch of dentistry that deals with the science and treatment of periodontal diseases. Synonym: *periodontics.*

22. *Periodontist.* One who is learned in the science of periodontology and skilled in the treatment of periodontal diseases.

23. *Periodontitis.* Inflammatory disease of the periodontium. *Periodontitis simplex*—sequela of chronic gingivitis. *Periodontitis complex* due to systemic disease or secondary to periodontosis. The condition may be further defined and described by such adjectives as acute, chronic, ulcerative, purulent (suppurative), etc.

24. *Periodontium.* The investing and supporting tissues surrounding the tooth, namely the periodontal membrane, the gingiva, and the alveolar bone.

25. *Periodontoclasia.* It is recommended that this term be discarded. The previous Nomenclature Committee recommended that this term be used to "indicate nonspecifically and collectively all destructive and degenerative diseases of the periodontium". However, the present Committee feels that the usage of a collective term for a disease complex is not only unjustified but confusing. Thanks to the progressive research of the past years, we can differentiate between different forms of periodontal disease which we can classify according to their pathology as inflammatory, degenerative, atrophic, and hyperplastic. These different pathologic changes manifest themselves with different clinical symptoms which should be recognized and expressed in a properly made diagnosis. If one speaks of "periodontoclasia" it is not clear what form of the disease complex is meant, but if one would refer to a certain case as an "acute hyperplastic scorbutic gingivitis" or "chronic tuberculous periodontitis" or "periodontosis" (early or late stage) everyone would understand. In the future, the tendency should be to separate more and more the different forms of periodontal disease. Only by separation into smaller units may we gain more insight into the pathologic and etiologic factors characteristic of the different groups of disturbances.

The soundness of this recommendation is obvious but it may be made more so by a comparison with medical terms. For example, one may speak of "heart disease" but only if the disease is specified to be an "endocarditis", "pericarditis", "myocarditis" or "valvular disease" and these diseases again specified as "rheumatic", "congenital", "syphilitic" and "acute" or "chronic", "bacterial" or "non-bacterial", etc., do we actually know the basic features of the disturbance.

The use of general terms which are not immediately recognized as such results in much confusion. It is recommended that as an important step forward, the term "periodontoclasia" be discarded and that we speak only in general terms of "periodontal diseases" and specify the condition under dis-

cussion, according to its basic pathologic, etiologic, and clinical features.

26. *Periodontology.* The science and study of the periodontium and periodontal diseases.

27. *Periodontosis.* Non-inflammatory degenerative destruction of the periodontium, characterized by degeneration of regular (functionally arranged) connective tissue elements of the periodontal membrane, leading to bone resorption, epithelial proliferation, migration of teeth, pocket formation, and ultimately exfoliation of teeth.

28. *Pocket. See—gingival pocket, periodontal pocket, intra-alveolar periodontal pocket.* (*See* diagram.)

29. *Traumatism.* Morbid condition of tissue caused by mechanical trauma (pressure necrosis and its consequences; thrombosis, hemorrhage, resorption of bone and cementum).

30. *Vincent's infection.* It is recommended that this designation of a certain type of gingival inflammation be discarded. The term was used to designate an acute ulcerative gingival inflammation. Properly we should speak of an "acute, sub-acute or chronic necrotizing ulcerative gingivitis". The term Vincent's angina should be discarded.

The Committee on Nomenclature takes this opportunity to express its sincere appreciation for the many valuable suggestions received from members of the Academy: Doctors Dickson G. Bell, F. E. Beube, Clarke E. Chamberlain, Edgar D. Coolidge, Henry M. Goldman, Raymond E. Johnson, Arthur H. Merritt, Grace Rogers Spalding, Samuel R. Parks, and especially Harry Sicher.

Clayton H. Gracey
Maynard K. Hine
Robert G. Kesel
Harry Lyons
Balint Orban, *Chairman*

Letter to the Editor

August 25, 1947

To the Editor of the Journal
of Periodontology:

The definition given for *Periodontosis* in paragraph 27 (Report of the Nomenclature Committee, *J. Periodont.* 18(3):112. July, 1947) is a perfect excerpt of my description of "Diffuse atrophy of the alveolar bone" (1923) with the only difference that the emphasis is not laid on the bone but on the periodontium, following the proposition of Henry M. Goldman (*J. Periodont.* 18:34. Jan. 1947).

I have felt since that the term chosen in 1923 was not adequate because the downgrowth of the epithelium at the base of the sulcus, which is such an important part of the entire picture, is not included. It was explained in the paper mentioned above that the disappearing of the bone is by no means a primary development, but a consequence of the deterioration of the cementum surface. The teaching of the "Biology of the Cementum" started with a paper of that title. I realized that a primary pathology of the bone is out of the question. As yet no dentist has ever observed a continuation of bone pathology after the tooth has been eliminated. After about a quarter of a century of study and research, I proposed the term "Cementopathia" in the January, 1946 issue of this Journal.

At the first glance it may seem unimportant whether we designate the bone or the periodontium as the main site of pathology. However, the difference between the "Diffuse Atrophy of the Alveolar Bone" and the new attempt to locate the pathology in the connective tissue which surrounds the tooth is very great. It appears that the attempt is being made to put the car of our concern on an entirely wrong track, and that would be fatal. Now an effort is made to locate the primary pathology in an independent change of the connective tissue and to designate the pathology of loosening and loss of the teeth as primarily beginning with a pathology of the connective tissue. Goldman is already investigating possibilities of general connective tissue deterioration. What I just stated applies here also. No one

as yet has observed a continuation of connective tissue disturbance after the tooth in question has been eliminated. I do not wish to be misunderstood. No one is entitled to regulate research. Goldman is qualified and entitled to back up his viewpoint as much as anyone else until the day a decision crystallizses. Free enterprise mixed with common sense and honesty is the foundation of research as well as of economics.

But it seems to me premature to base the decision of a nomenclature committee on a single paper which the cautious author (Goldman) himself entitles as being a "preliminary report". The Academy has previously used the term "periodontoclasia", giving this name a broad interpretation. The committee dropped this name on account of this broad interpretation. The dropping of the broad interpretation appears justified. But I see no reason why the name could not be retained with the explanation given to the word "Periodontist". Both names designate that something is wrong around the tooth that is not inflammatory. But this jumping around with names is not desirable. The nomenclature committee of the Academy should not use the content of a preliminary report as a basis for nomenclature.

Faithfully yours,
B. Gottlieb

Announcements

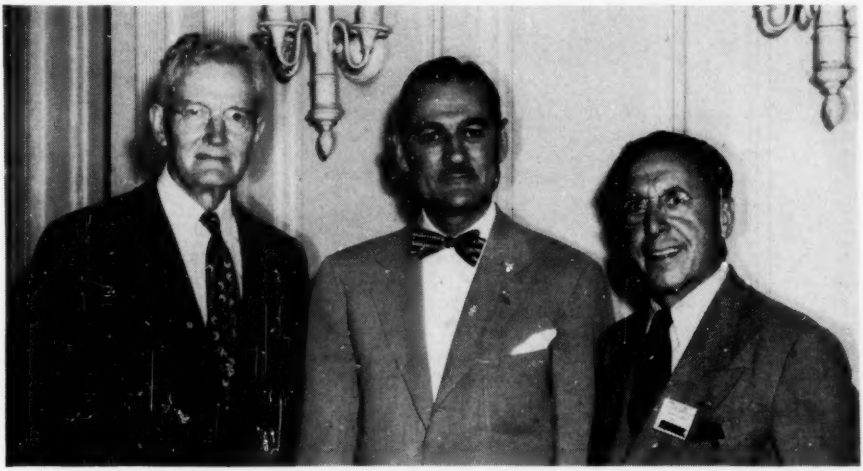
The American Academy of Dental Medicine will hold a luncheon meeting prior to the Greater New York Dental Meeting at the Hotel Pennsylvania on Sunday, December 7th, 1947 at 12:30 P.M. Round Table Discussion will follow. All members and interested dentists and physicians are invited to attend. At a recent meeting the following officers were elected for the year 1947-1948. Dr. Sidney Sorrin, *President;* Dr. J. Lewis Blass, *President-elect;* Dr. William M. Greenhut, *Secretary;* Dr. George A. Bruns, *Treasurer;* Dr. Allan N. Arvins, *Editor.* Address all communications to Dr. William M. Greenhut, *Secretary,* 124 East 84th Street, New York City.

"In Our Opinion Discussion in the January, 1948 issue—Subject: "Is Reattachment or Pocket Elimination Essential to Retaining Periodontally Affected Teeth in Health?"

A Brief Report of the Thirty-first Annual Meeting of the American Academy of Periodontology

By MILDRED W. DICKERSON, D.D.S., *Washington, D. C.*

The thirty-first annual meeting of the American Academy of Periodontology was held at the Copley Plaza Hotel, in Boston, July 31, August 1 and 2, just prior to the annual meeting of the American Dental Association, as has been the custom, with few exceptions, since 1917. The registered attendance was 254 and since the Tenth International Dental Congress was meeting in Boston at this time, eighteen of the guests were from foreign countries.



Left to right: Edgar D. Coolidge, *Past-President;* Raym ond E. Johnson, *President;* Edward L. Ball President-Elect.

The morning session of July 31 was given over to the annual reports of the officers and committees and closed with the splendid address of President Edgar D. Coolidge in which progressive action in several directions was recommended.

The Committee on Program with Dr. Dickson G. Bell as Chairman, together with the Committee on Scientific Investigation under Dr. Frank E. Beube, provided a most interesting and scientific program covering a wide variety of subjects of importance to periodontists.

The afternoon session opened with a discussion of "Endocrine Disturbances Related to Gingival Pathology". by Dr. Daniel E. Ziskin. His subject matter and slides included some rarely seen types of cases.

Dr. Thomas J. Cook followed with a full discussion of "The Blood Dyscrasias in Relation to Periodontal Disturbances with Special Reference to the Leukemias". The third essayist, Dr. William G. McIntosh, of Toronto, in his paper on the "Relation of Ascorbic Acid Intake to Gingivitis" recounted some very interesting and practical observations that were made in a dietary study on 150 members of the Royal Canadian Air Force over a five month period. A report of this study was published previously in the Journal of the Canadian Dental Association.

The Committee on Scientific Investigation had charge of the evening session and presented a program on the subject of diabetes. The first essayist, Elliott P. Joslin, M.D. spoke on "The Coöperative Relationship of Dentists and Physicians on the Diabetic Problem". He was followed by Alexander Marble, M.D. and Herbert Pollack, M.D. in a Symposium: "Diabetes Mellitus, Its Rela-

Left to right: J. C. Westbrook, Jr., *Chairman*, Clinic Committee; C. B. Vaughan, *Chairman* Local Arrangements; Roy O. Elam, Member of Council.

tion to Oral Pathosis". Dr. Daniel E. Ziskin was the formal discusser.

On Friday morning, August 1, the Committee on Scientific Investigation continued by presenting S. Burt Wolback, M.D., Stephen Hudack, M.D., and Joseph C. Aub, M.D., in a Symposium: "What are the Various Factors Which Affect the Healing of Bone after Injury and Infection?" The slides shown during these talks were most excellent. Dr. Harry Sicher, the discusser and an authority on the subject of bone, brought out some most important information. A lively informal discussion followed. It was noted throughout the scientific program that all of the essayists made some practical application of the material presented.

For many years the Round Table Luncheon has been one of the interesting and enjoyable features of the Academy program. This year Drs. Harold G. Ray and Howard A. Hartman adopted the plan of having fewer, but larger tables in order to shorten the period devoted to the reports. The idea of fewer reports was good, but the tables were so large one could not hear the Leader and missed much of the discussion. They sent out, in advance, the ten well chosen subjects to be discussed and asked the members to register by mail for the subject of their choice.' The latter idea is to be especially commended.

The women guests of the Academy, always so welcome, were seated at one large table over which Mrs. Edgar D. Coolidge very graciously presided. The Luncheon program closed with her talk on "Meeting Dentists and Dental Meetings in the U.S., Canada, and Europe". She narrated many an interesting incident she had experienced when she accompanied Dr. Coolidge to various meetings at home and abroad.

Following the luncheon ten outstanding clinics were given under the chairmanship of Dr. Westbrook. Both he and the clinicians are to be congratulated. Each of the clinics was deserving of more time and

Left to right: James E. Aiguier, *Chairman* Entertainment Committee; Samuel R. Parks, *Chairman* Executive Council; Robert L. Dement, Council Member. (Photographs—*Courtesy of* Howard A. Hartman.)

better space assignment so that the members might have had the opportunity of hearing and seeing all of them.

On Saturday morning, August 2, three scientific papers were scheduled. The first one, "Stomatitis and Paradentopathies" by René Jaccard of Geneva, Switzerland, was read by Prof. O. Müller substituting for Dr. Jaccard who was unable to be present. The second one "Inflammation and its Various Implications" was presented by Valy Menkin, M.D., author of *Dynamics of Inflammation*. The third paper "Concerning the Pathogenesis of Periodontal Disease" by Dr. Harold K. Box, was read by Dr. Charles H. Williams, Dr. Box being unable to present the paper himself.

The meeting closed with a short business session and the election of officers. Dr. Raymond E. Johnson, President-Elect, was installed as President, Dr. Edward L. Ball is the new President-Elect. Dr. Clarke E. Chamberlain was re-elected Secretary and Dr. C. Wesley Marriott was re-elected Treasurer.

The social features of the meeting were two—a well-attended formal banquet on Friday evening with music, and Bill Cunningham, International News Commentator, as the principal speaker, and an informal Tea for members and guests who were entertained by a "Continent in Color" presented by Dr. Howard A. Hartman.

All who contributed to the success of the Thirty-first Annual Meeting deserve and have the sincere appreciation of those in attendance.

Special Announcement to Subscribers

Please note that the subscription price of the Journal of Periodontology will be advanced to $6.00 January 1, 1948. If your renewal is received *before* that date it will be accepted at the present rate of $4.00.

Subscription Manager
E. ROMLE ROMINE
David Broderick Bldg.
Detroit 26, Michigan

BOOK REVIEWS

Journal of the History of Medicine and Allied Sciences. A quarterly. New York; Henry Schuman. Subscription rate $7.50 a year; single copies $2.50.

Henry Schuman, antiquarian bookseller and publisher, who has specialized in books on medical history for more than a dozen years, has added this new quarterly to the already impressive list of publications associated with his imprint. The only other American journal in this field at present is the *Bulletin of the History of Medicine,* and since the discontinuance of the quarterly *Annals of Medical History* in 1942 there has been need of additional outlets for scholarly historical studies. Perusal of the first volume of this new journal, now complete, leads one to believe that the *Annals* has a worthy successor.

Editor George Rosen has both the professional training and the experience to qualify him for his position. Among his assistants are many names long prominent as medical historians.

During the first year the *Journal of the History of Medicine and Allied Sciences* has made good its promise of publishing "work relating to all aspects of the history of medicine, public health, dentistry, nursing, pharmacy, veterinary medicine and the various sciences that impinge on medicine".

The October 1946 issue is one that many persons will want to own even if they do not become regular subscribers. It is the anesthesia centennial number, and its twenty-two articles cover the history of anesthesia in all its phases and in all parts of the world. Thomas E. Keys, Noel A. Gillespie, Edward H. Hume and Chauncey D. Leake are among the contributors who add authority and distinction to this collection.

Each issue has averaged almost two hundred pages of a mat paper of good quality. The format in simple good taste gives the journal an appearance of easy readibility.—*Murrell C. Wellman.*

Reprinted from J.A.D.A. 35(5):382. Sept. 1, 1947

THE JOURNAL OF APPLIED NUTRITION

The official publication of the American Academy of Applied Nutrition is published quarterly. It is now in its second issue.

For further information write

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Send your 1948 subscription for the Journal of Periodontology in now. For the reason—See p. 181.—ED.

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